Multiple Brain Abscesses and Tetraventricular Hydrocephalus Complicating Enterobacter cloacae Sepsis in a Premature Infant: A Case Report and Literature Review

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DOI: 10.36347/sjmcr.2021.v09i06.014 | Received: 10.04.2021 | Accepted: 16.05.2021 | Published: 21.06.2021

Abstract

Enterobacter cloacae, a facultative anaerobic Gram-negative bacterium, is part of human enteric flora. However, it has gained clinical importance as it has emerged as a nosocomial pathogen, especially in neonatal intensive care units [1, 2]. Enterobacter cloacae complex comprises six heterogenic species which act as pathogens when travel to other parts of the body [1]. Enterobacter cloacae may cause nosocomial infections including respiratory (pneumonia) and urinary tract infections, skin infection, intra-abdominal infection, endocarditis, osteomyelitis, and septic arthritis [3]. Additionally, this pathogen can contaminate medical devices or surgical instruments resulting in nosocomial outbreaks [1]. The major problem with Enterobacter cloacae is its difficult treatment as it is resistant to various antibiotic agents including penicillins, and cephalosporins (3rd generation) [4]. This resistance of Enterobacter cloacae to various antibiotics agents owes to the production of certain enzymes e.g., chromosomal AmpC β-lactamases (cAmpC) [4]. Mortality rate of blood stream infections (BSIs) caused by Enterobacter cloacae ranges from 27% to 61% [5]. Although BSIs caused by Enterobacter cloacae are uncommon; however, their high mortality rate warrants careful assessment of the infection in-time. Brain abscess is one of the rare complications of Enterobacter cloacae infection [6]. Unfortunately, this pathogen can cause brain abscess in immune-competent children [7]. In this case report, we present a premature male infant who developed multiple brain abscesses and tetraventricular hydrocephalus following Enterobacter cloacae sepsis.

CASE PRESENTATION

A newborn male baby was hospitalized in neonatal intensive care unit for respiratory distress. The baby was born to a 29-year-old mother (gravida 4, para 3, miscarriage 1) who had 3 alive children. The poorly monitored pregnancy was estimated at 35 weeks according to the Farr score when she presented with greenish fetid leucorrhoea during the 3rd trimester. The blood group of the mother was AB positive. Although the fetus had breech presentation, however the baby was delivered via spontaneous vaginal delivery. At birth, Apgar score was 10/10 which decreased to 5/10 after 5 minutes. The respiratory symptoms started at the first hour of life.

On the admission examination, newborn was pink in appearance, not very reactive and was gesturing a little. Heart rate was 138 beats/min, temperature was at 35.7, respiratory rate was 62 cycles/min, and SaO2 was 92% in the open air. The baby had weight of 2.3 kg, height of 43 cm, and head circumference of 31 cm. Neurological examination showed axial hypotonia with...
good peripheral tone and sucking reflex. Pleuropulmonary examination showed respiratory distress at Silverman score of 6/10. Biological assessment of the baby was normal and there were no congenital anomalies.

The baby was offered bottle feeding and gavage, antibiotic therapy (amoxicillin and gentamicin) and injection vitamin K. Later, the baby’s clinical (fever, mottling and jaundice) and biological conditions got worsened. CRP value was at 40 mg/l. A blood culture performed returned positive showing sepsis for Enterobacter cloacae extended spectrum betalactamase strain (nosocomial infection). The newborn was put on imipenem and amikacin. The baby’s condition improved and was discharged after 10 days of hospitalization.

After one month, the newborn presented with fever at 40 °C with disturbed consciousness. A brain scan was obtained which showed a passive quadriventricular hydrocephalus without detectable obstacle (Figure 1), with multiple lesions related to cerebral abscesses (Figure 2), some of which are compressive, associated with an aspect of ventriculitis.

The analysis of cerebrospinal fluid revealed a cloudy appearance with proteinorachia at 2.14 g/L and glycorrhachia at 0.03 g/L. The culture was positive for the extended spectrum Enterobacter cloacae strain betalactamase. Diagnosis of Enterobacter cloacae meningitis, complicated by brain abscesses and tetraventricular hydrocephalus has been confirmed. The patient underwent surgical drainage of the abscesses with placement of an external bypass drain, and was put on imipenem and amikacin parenterally, as well as amikacin and colistin intrathecally. The patient’s clinical and biological status were significantly improved within 15 days of treatment.

**DISCUSSION**

Although rare, but Enterobacter cloacae sepsis may lead to life-threatening infection meningitis, brain abscesses and hydrocephalus in premature infants. A few reports have been published on Enterobacter sepsis, meningitis, brain abscess and hydrocephalus. In the present case, a premature infant suffered from Enterobacter cloacae meningitis, complicated by brain abscesses and tetraventricular hydrocephalus which required aggressive course of broad-spectrum antibiotics. Therefore, the infants with infections difficult-to-treat should be considered for blood culture to rule out Enterobacter cloacae sepsis so that life-threatening cerebral infection can be prevented. Unfortunately, Enterobacter cloacae can infect both immune-competent and immunocompromised people.

Traore et al., [6] presented a 21-days premature infant who developed multiple brain abscesses following nosocomial infection with Enterobacter cloacae. Saini et al., [7] reported an immune-competent 10-year-old boy with bilateral multiple pyogenic brain abscesses caused by Enterobacter cloacae. They confirmed the presence of pathogen by culturing the aspirated material. They offered intravenous antibiotic (imipenem) for 18 weeks to resolve the infection. They suggested early surgical drainage, appropriate antibiotic therapy and patient tailored duration of treatment in such complicated infections. Enterobacter cloacae can contaminate medical and surgical instruments leading to nosocomial outbreaks. Various outbreaks out Enterobacter cloacae has been reported in the literature. Van Nierop et al., [8] reported an outbreak in a neonatal ICU with nine deaths, and Kuboyama et al., [9] reported three outbreaks having 42 systemic infections with 34% mortality rate. Similarly in Netherland, van den Berg et al., [10] reported an outbreak of multi-resistant

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**Fig-1:** Sagittal section of a brain CT scan with injection of contrast product, which shows a hypodense intraparenchymal collection with peripheral enhancement related to the abscess

**Fig-2:** Axial section of a cerebral CT scan with injection of contrast product, showing 2 frontal and parietal collections, with tetraventricular hydrocephalus

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Enterobacter cloacae infection in a neonatal ICU of the University Hospital of Nijmegen due to contaminated thermometers. Later, adequate disinfection of thermometers put an end to the outbreak.

CONCLUSION

Enterobacter cloacae may cause lethal cerebral infection requiring invasive surgical interventions and prolonged antibiotic therapy while having high risk of mortality. Therefore, implementation of proper disinfection measures, early detection of the infection and its management will prevent lethal complications caused by Enterobacter cloacae.

REFERENCES